WEST Search History

Hide Items | Restore | Clear | Cancel

DATE: Tuesday, April 11, 2006

Hide?	Set Name	Query	Hit Count
	DB=PGPB; THES=ASSIGNEE; PLUR=YES; OP=ADJ		
☐	L19	L18 and HMGB$	2
☐	L18	newman-walter.in.	16
☐	L17	20040156851.pn.	1
	DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ		
☐	L16	20040156851.pn.	2
	DB=USPT,USOC,EPAB,JPAB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ		
☐	L15	6448223.pn.	2
☐	L14	L10 same effective	8
☐	L13	L10 same additive	0
☐	L12	L10 same synerg$	1
☐	L11	L10 same advanta$	1
☐	L10	L6 same (sepsis or arthritis)	114
☐	L9	L6 same (sepsis same arthritis)	1
☐	L8	L6 same (sepsis with arthritis)	0
☐	L7	L6 same (combination adj therapy)	4
☐	L6	infliximab or etanercept or adalimumab or (cdp adj (870 or 571)) or lenercept	317
☐	L5	L3 same (combination adj therapy)	35
☐	L4	L3 same combination	294
☐	L3	L2 same l1	3749
☐	L2	TNF$	21633
☐	L1	sepsis or arthritis	63571

END OF SEARCH HISTORY

United States Patent and Trademark Office

Trademarks > Trademark Electronic Search System(Tess)

TESS was last updated on Tue Apr 11 04:16:15 EDT 2006

Logout Please logout when you are done to release system resources allocated for you.

Start List At: OR Jump to record: **Record 1 out of 4**

TARR Status | ASSIGN Status | TDR | TTAB Status *(Use the "Back" button of the Internet Browser to return to TESS)*

Word Mark	ENBREL **ETANERCEPT**
Goods and Services	IC 009. US 021 023 026 036 038. G & S: Prerecorded audio tapes and discs, videotapes and discs, and compact discs featuring information relating to human immune diseases and conditions. FIRST USE: 20000800. FIRST USE IN COMMERCE: 20000800
Mark Drawing Code	(3) DESIGN PLUS WORDS, LETTERS, AND/OR NUMBERS
Design Search Code	02.01.33 - Men, grotesque; Monsters (not robots); Snowmen; Stick figures 26.11.20 - Rectangles inside one another 26.11.21 - Rectangles that are completely or partially shaded
Serial Number	75643724
Filing Date	February 19, 1999
Current Filing Basis	1A
Original Filing Basis	1B
Published for Opposition	October 5, 1999
Registration Number	2518007
Registration Date	December 11, 2001
Owner	(REGISTRANT) Immunex Corporation CORPORATION WASHINGTON 51 University Street Seattle WASHINGTON 98101

Attorney of Record	LAURENCE R. HEFTER
Prior Registrations	2220795
Disclaimer	NO CLAIM IS MADE TO THE EXCLUSIVE RIGHT TO USE "ETANERCEPT" APART FROM THE MARK AS SHOWN
Description of Mark	The mark consists of the word "ENBREL" a rectangle and the stylized figure of a person in motion.
Type of Mark	TRADEMARK
Register	PRINCIPAL
Live/Dead Indicator	LIVE

```
=> d his

     (FILE 'HOME' ENTERED AT 06:55:17 ON 11 APR 2006)

     FILE 'DISSABS, 1MOBILITY, AGRICOLA, AQUASCI, BIOTECHNO, COMPENDEX,
     COMPUAB, CONF, CONFSCI, ELCOM, HEALSAFE, IMSDRUGCONF, LIFESCI, OCEAN,
     PAPERCHEM2, PASCAL, POLLUAB, SOLIDSTATE, ADISCTI, ADISINSIGHT, ADISNEWS,
     ANABSTR, ANTE, AQUALINE, BIOENG, BIOSIS, ...' ENTERED AT 06:55:26 ON 11
     APR 2006
L1         2942 S HMG? (S) (CYTOKINE OR TNF)
L2         1599 S L1 (S) TNF
L3          686 S L2 (S) (ANTI OR ANTIBOD? OR MAB)
L4          549 DUP REM L3 (137 DUPLICATES REMOVED)

     FILE 'DISSABS, 1MOBILITY, AGRICOLA, AQUASCI, BIOTECHNO, COMPENDEX,
     COMPUAB, CONF, CONFSCI, ELCOM, HEALSAFE, IMSDRUGCONF, LIFESCI, OCEAN,
     PAPERCHEM2, PASCAL, POLLUAB, SOLIDSTATE, ADISCTI, ADISINSIGHT, ADISNEWS,
     ANABSTR, ANTE, AQUALINE, BIOENG, BIOSIS, ...' ENTERED AT 07:05:32 ON 11
     APR 2006

     FILE 'STNGUIDE' ENTERED AT 07:32:57 ON 11 APR 2006

     FILE 'DISSABS, BIOTECHNO, LIFESCI, PASCAL, ADISCTI, BIOTECHDS, CAPLUS,
     DGENE, DRUGU, EMBAL, EMBASE, ESBIOBASE, FEDRIP, GENBANK, IFIPAT,
     JICST-EPLUS, MEDLINE, PHARMAML, PHIC, PHIN, PROMT, TOXCENTER, USPATFULL,
     USPAT2, WPIDS, NLDB' ENTERED AT 07:40:00 ON 11 APR 2006

     FILE 'STNGUIDE' ENTERED AT 07:45:30 ON 11 APR 2006

     FILE 'DISSABS, BIOTECHNO, LIFESCI, PASCAL, ADISCTI, BIOTECHDS, CAPLUS,
     DGENE, DRUGU, EMBAL, EMBASE, ESBIOBASE, FEDRIP, GENBANK, IFIPAT,
     JICST-EPLUS, MEDLINE, PHARMAML, PHIC, PHIN, PROMT, TOXCENTER, USPATFULL,
     USPAT2, WPIDS, NLDB' ENTERED AT 07:48:02 ON 11 APR 2006

     FILE 'STNGUIDE' ENTERED AT 07:48:10 ON 11 APR 2006

     FILE 'DISSABS, 1MOBILITY, AGRICOLA, AQUASCI, BIOTECHNO, COMPENDEX,
     COMPUAB, CONF, CONFSCI, ELCOM, HEALSAFE, IMSDRUGCONF, LIFESCI, OCEAN,
     PAPERCHEM2, PASCAL, POLLUAB, SOLIDSTATE, ADISCTI, ADISINSIGHT, ADISNEWS,
     ANABSTR, ANTE, AQUALINE, BIOENG, BIOSIS, ...' ENTERED AT 07:48:34 ON 11
     APR 2006
L5           129 S L4 (S) HMGB?
L6           129 DUP REM L5 (0 DUPLICATES REMOVED)
```

```
=> d his

     (FILE 'HOME' ENTERED AT 06:55:17 ON 11 APR 2006)

     FILE 'DISSABS, 1MOBILITY, AGRICOLA, AQUASCI, BIOTECHNO, COMPENDEX,
     COMPUAB, CONF, CONFSCI, ELCOM, HEALSAFE, IMSDRUGCONF, LIFESCI, OCEAN,
     PAPERCHEM2, PASCAL, POLLUAB, SOLIDSTATE, ADISCTI, ADISINSIGHT, ADISNEWS,
     ANABSTR, ANTE, AQUALINE, BIOENG, BIOSIS, ...' ENTERED AT 06:55:26 ON 11
     APR 2006
L1         2942 S HMG? (S) (CYTOKINE OR TNF)
L2         1599 S L1 (S) TNF
L3          686 S L2 (S) (ANTI OR ANTIBOD? OR MAB)
L4          549 DUP REM L3 (137 DUPLICATES REMOVED)

     FILE 'DISSABS, 1MOBILITY, AGRICOLA, AQUASCI, BIOTECHNO, COMPENDEX,
     COMPUAB, CONF, CONFSCI, ELCOM, HEALSAFE, IMSDRUGCONF, LIFESCI, OCEAN,
     PAPERCHEM2, PASCAL, POLLUAB, SOLIDSTATE, ADISCTI, ADISINSIGHT, ADISNEWS,
     ANABSTR, ANTE, AQUALINE, BIOENG, BIOSIS, ...' ENTERED AT 07:05:32 ON 11
     APR 2006

     FILE 'STNGUIDE' ENTERED AT 07:32:57 ON 11 APR 2006

     FILE 'DISSABS, BIOTECHNO, LIFESCI, PASCAL, ADISCTI, BIOTECHDS, CAPLUS,
     DGENE, DRUGU, EMBAL, EMBASE, ESBIOBASE, FEDRIP, GENBANK, IFIPAT,
     JICST-EPLUS, MEDLINE, PHARMAML, PHIC, PHIN, PROMT, TOXCENTER, USPATFULL,
     USPAT2, WPIDS, NLDB' ENTERED AT 07:40:00 ON 11 APR 2006

     FILE 'STNGUIDE' ENTERED AT 07:45:30 ON 11 APR 2006

     FILE 'DISSABS, BIOTECHNO, LIFESCI, PASCAL, ADISCTI, BIOTECHDS, CAPLUS,
     DGENE, DRUGU, EMBAL, EMBASE, ESBIOBASE, FEDRIP, GENBANK, IFIPAT,
     JICST-EPLUS, MEDLINE, PHARMAML, PHIC, PHIN, PROMT, TOXCENTER, USPATFULL,
     USPAT2, WPIDS, NLDB' ENTERED AT 07:48:02 ON 11 APR 2006

     FILE 'STNGUIDE' ENTERED AT 07:48:10 ON 11 APR 2006
```

L4 ANSWER 541 OF 549 DGENE COPYRIGHT 2006 The Thomson Corp on STN
ACCESSION NUMBER: AAA88303 DNA DGENE
TITLE: Novel pharmaceutical compositions used to treat conditions characterized by activation of the inflammatory cytokine cascade, especially sepsis, or to cause weight loss and treat obesity -
INVENTOR: Tracey K J; Wang H
PATENT ASSIGNEE: (PICO-N)PICOWER INST MEDICAL RES.
PATENT INFO: WO 2000047104 A2 20000817 35
APPLICATION INFO: WO 2000-US3583 20000211
PRIORITY INFO: US 1999-248574 19990211
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: 2000-549070 [50]
DESCRIPTION: High mobility group 1 PCR primer SEQ ID NO:2.
AB The present invention describes a pharmaceutical composition for treating conditions characterised by activation of the inflammatory **cytokine** cascade, comprising a high mobility group (**HMG**)1 antagonist or inhibitor. The **HMG1** antagonist containing compositions are used to treat conditions characterised by activation of the inflammatory **cytokine** cascade, especially sepsis. An antagonist of an early sepsis mediator, especially an antagonist of tumour necrosis factor (**TNF**), interleukin (IL)-1beta, MIF, or IL-6, an **antibody** to **TNF**, or MIF, or an IL-1 receptor antagonist, may also be administered to enhance the sepsis treatment. **HMG1** can be used to cause weight loss and treat obesity. The present invention also describes methods which can be used to diagnose and prognose the severity, or likely clinical course, of sepsis in patients exhibiting shock-like symptoms or at risk of exhibiting symptoms associated with inflammatory cascade mediated conditions. The diagnostic method is applied to serum, or other tissues or fluids such as cerebrospinal fluid or urine. The present sequence represents a PCR primer for **HMG1**, which is used in an example from the present invention.
AB The present invention describes a pharmaceutical composition for treating conditions characterised by activation of the inflammatory **cytokine** cascade, comprising a high mobility group (**HMG**)1 antagonist or inhibitor. The **HMG1** antagonist containing compositions are used to treat conditions characterised by activation of the inflammatory **cytokine** cascade, especially sepsis. An antagonist of an early sepsis mediator, especially an antagonist of tumour necrosis factor (**TNF**), interleukin (IL)-1beta, MIF, or IL-6, an **antibody** to **TNF**, or MIF, or an IL-1 receptor antagonist, may also be administered to enhance the sepsis treatment. **HMG1** can be used to cause weight loss and treat obesity. The present invention also describes methods which can be used. . . serum, or other tissues or fluids such as cerebrospinal fluid or urine. The present sequence represents a PCR primer for **HMG1**, which is used in an example from the present invention.

ANSWER 65 OF 129 DRUGU COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2003-01232 DRUGU P
TITLE: HMGB1-targeted therapy ameliorates collagen-induced arthritis in mice.
AUTHOR: Kokkola R M J; Sundberg E; Tracey K J; Andersson U; Harris H E
CORPORATE SOURCE: Karolinska-Inst.
LOCATION: Stockholm, Swed.; Manhasset, N.Y., USA
SOURCE: Arthritis Rheum. (46, No. 9, Suppl., S566, 2002)
CODEN: ARHEAW ISSN: 0004-3591
AVAIL. OF DOC.: Karolinska Institutet, Stockholm, Sweden.
LANGUAGE: English
DOCUMENT TYPE: Journal
FIELD AVAIL.: AB; LA; CT
FILE SEGMENT: Literature

Arthritis CIA model

AB The effects of i.p. high mobility group box chromosomal protein 1 (HMGB1) A-box and anti-HMGB1 antibodies were investigated in an in-vitro study and in in-vivo studies in mice with collagen-induced CIA. HMGB1 A-box decreased production of proinflammatory cytokines induced by HMGB1 B-box. HMGB1 A-box and anti-HMGB1 antibodies ameliorated arthritis in-vivo in mice. In conclusion, the results show HMGB1 may be a future target for therapy of human arthritis an A-box and/or anti-HMGB1 antibodies could be sued as antagonists of excessive cytokine production in arthritis. (conference abstract: American College of Rheumatology 66th Annual Scientific Meeting and the Association of Rheumatology Health Professionals 37th Annual Scientific Meeting, New Orleans, Louisiana, USA, 2002).

ABEX. . . Methods DBA1/J mice were immunized with bovine collagen type II to induce CIA and were boosted on day 21, I.p. **HMGB1** A-box or **anti-HMGB1 antibodies** were given for 7 days. Results In-vitro in mouse peritoneal macrophages, **HMGB1** B-box-induced **TNF** production was decreased after preincubation with **HMGB1** A-box. In-vivo, **HMGB1** A-box or **anti-HMGB1 antibodies** treated mice showed lower mean arthritis indexes compared with controls. The number of affected paws and paws with maximal arthritis. . .

L6 ANSWER 62 OF 129 PASCAL COPYRIGHT 2006 INIST-CNRS. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 2004-0306505 PASCAL

TITLE (IN ENGLISH): High mobility group box-1 as a therapeutic target downstream of tumor necrosis factor
First Annual Cambridge Colloquium on Genetic, Molecular, and Cellular Basis of Innate Immunity and Sepsis

AUTHOR: CZURA Christopher J.; HUAN YANG; TRACEY Kevin J.
ABRAHAM Edward (ed.); CALANDRA Thierry (ed.)

CORPORATE SOURCE: Laboratory of Biomedical Science, North Shore-Long Island Jewish Research Institute, Manhasset, New York, United States
Division of Pulmonary Sciences and Critical Care Medicine, University of Colorado, Denver, United States; Department of Internal Medicine, Division of Infectious Diseases, Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland
The International Sepsis Forum, INC (patr.)

SOURCE: The Journal of infectious diseases, (2003), 187(SUP2), S391-S396, 48 refs.
Conference: 1 Annual Cambridge Colloquium on Genetic, Molecular, and Cellular Basis of Innate Immunity and Sepsis, Cambridge (United Kingdom), 14 Jul 2002
ISSN: 0022-1899 CODEN: JIDIAQ

DOCUMENT TYPE: Journal; Conference
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-2052, 354000118565400130

AB The discovery of tumor necrosis factor (**TNF**) as a necessary and sufficient mediator of systemic inflammation started a new field of research to rationally modulate **cytokine** responses to therapeutic advantage. However, the early kinetics of the **TNF** response during infection defined an extremely narrow window of opportunity during which **anti-TNF** therapeutics are efficacious, hampering clinical development for severe sepsis. Because death from severe sepsis often occurs as a late phenomenon, we began a search began for putative "late" mediators that could be targeted after the onset of infection. We have now identified high mobility group box-1 (**HMGB1**) as a late mediator of endotoxemia and sepsis. **HMGB1** is released by activated macrophages, induces the release of other proinflammatory mediators, and mediates lethality when overexpressed. Administration of **anti-HMGB1** **antibodies** inhibit systemic inflammation, even in established cases, because **HMGB1** activity is elevated at significantly later time points than **TNF** or interleukin-1. It will now be important to determine whether this wider window of activity can be translated into therapeutic advantage for human inflammatory disease.

AB The discovery of tumor necrosis factor (**TNF**) as a necessary and sufficient mediator of systemic inflammation started a new field of research to rationally modulate **cytokine** responses to therapeutic advantage. However, the early kinetics of the **TNF** response during infection defined an extremely narrow window of opportunity during which **anti-TNF** therapeutics are efficacious, hampering clinical development for severe sepsis. Because death from severe sepsis often occurs as a late phenomenon,. . . putative "late" mediators that could be targeted after the onset of infection. We have now identified high mobility group box-1 (**HMGB1**) as a late mediator of endotoxemia and sepsis. **HMGB1** is released by activated macrophages, induces the release of other proinflammatory mediators, and mediates lethality when overexpressed. Administration of **anti-HMGB1** **antibodies** inhibit systemic inflammation, even in established cases, because **HMGB1** activity is elevated at significantly later time points than **TNF** or interleukin-1. It will now be